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L1: Entry 1 of 1 File: USPT Dec 1, 1998

US-PAT-NO: 5843436
DOCUMENT-IDENTIFIER: US 5843436 A

TITLE: Method of preventing and treating bacterial infection of sutures and prosthetic devices, and promoting ingress of leukocytes into tumor foci

DATE-ISSUED: December 1, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Loike; John	Jamaica Estates	NY		
Silverstein; Samuel C.	New York	NY		

US-CL-CURRENT: 424/94.64; 424/423, 424/532, 424/94.63, 514/2

CLAIMS:

What is claimed is:

1. A method of preventing a chronic infection from occurring due to the presence of bacterial cells on a surface of a foreign body in a subject, which consists essentially of coating the foreign body before placing it in the subject with a fibrinolytic agent capable of preventing the accumulation of fibrin on the surface of the foreign body so as to permit leukocyte cells to reach and kill any bacterial cells present on the surface of the foreign body and thereby prevent the chronic infection.

2. The method of claim 1, wherein the foreign body is a prosthetic device.

3. The method of claim 1, wherein the foreign body is a catheter.

4. The method of claim 1, wherein the foreign body is a suture.

5. The method of claim 1, wherein the subject is a mammal.

6. The method of claim 5, wherein the mammal is a human.

7. The method of claim 1, wherein the fibrinolytic agent is a plasminogen activator.

8. The method of claim 7, wherein the plasminogen activator is urokinase.

9. The method of claim 7, wherein the plasminogen activator is streptokinase.

10. The method of claim 7, wherein the plasminogen activator is tissue plasminogen activator.

L7 ANSWER 1 OF 2 CANCERLIT
ACCESSION NUMBER: 96605559 CANCERLIT
DOCUMENT NUMBER: 96605559
TITLE: Biphasic effect (stimulation and suppression) by **tenascin** on human glioma cell migration (Meeting abstract).
AUTHOR: Berens M E; Giese A
CORPORATE SOURCE: Neuro-Oncology Lab., Barrow Neurological Inst. of St. Joseph's Hosp. and Medical Center, Phoenix, AZ 85013-4496.
SOURCE: J Cell Biochem, (1995) Suppl 19B 18.
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AB **Tenascin** is an extracellular matrix protein which is expressed in human gliomas. Cell receptors for **tenascin** are reported to utilize the alpha v subunit integrin as one chain of the heterodimer receptor. We tested whether purified **tenascin**, passively deposited on monolayer surfaces, influenced the adhesion or migration behavior of human glioma-derived cells, SF767. Studies of other ECM proteins (laminin, collagen, fibronectin, vitronectin) demonstrated that adhesion increases in a dose-dependent manner, with optimal (maximum) specific attachment by 30-60 minutes at 37 C using 100 ug/ml. In contrast, glioma adhesion to **tenascin** increased to a maximum degree at 10 ug/ml, but steadily decreased using coating concentrations of 33 and 100 ug/ml. Cell adhesion to **tenascin** could be completely blocked (to basal levels) using anti-**beta1 antibodies**.
Surprisingly, treatment with anti-alpha v antibodies led to slightly enhanced cell adhesion. Using a microliter scale migration assay (Berens et al, Clin Exp Mets; 1994) it was found that migration of glioma cells on **tenascin** was dose-dependently stimulated at coating concentrations of 1 and 3 ug/ml but cell migration was actually suppressed (to rates below that seen on BSA) when tested on 30 or 100 ug/ml. Migration on optimal concentrations of **tenascin** could be reversibly inhibited by treatment with anti-**beta1 antibodies**; treatment with anti-alpha v antibodies actually stimulated glioma migration. We conclude that glioma cells express two separate receptors for **tenascin**; and that ligand density, determined by different coating concentrations of **tenascin**, activates these different integrins.
The beta1 containing integrin(s) mediate adhesive and migratory responses, while the alpha v-containing integrin(s) appear to be counteradhesive and inhibitory to migration. These findings highlight the interplay between different integrins which recognize the same ECM protein, and demonstrate that the net response of a cell to complex extracellular matrix ligands is an integrated manifestation of differing, and possibly opposing, integrin-mediated reactions.